PB-237 793-T

TETRACARBONYLNICKEL INTOXICATION BY INHALATION

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Environmental Protection Agency
Research Triangle Park, North Carolina

September 1972

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EPA - TR. 121-73

PB 237 793-T

APTIC 48037

TETRACARBONYLNICKEL INTOXICATION BY INHALATION
[Inhalacni intoxikace tetrakarbonylniklem]

by

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Published in Pracovni lekarstvi [Occupational Medicine]
Vol 12, No 2, 1960, pp. 84-86.

Tetracarbonylnickel Ni $[CO]_4$ is a colorless liquid which smells like unburned coal [9], with boiling point 43.3°C., which is soluble in organic solutions, formed by the action of carbon monoxide on metallic nickel, and is most often used for nickel refinement.

Industrial tetracarbonylnickel intoxications are relatively rare. Except for a single mass intoxication in a British factory [4], in the majority of cases we are dealing with isolated, random, accidental, acute intoxications which occur most often among laboratory workers [10].

In our literature we found no reference to industrial nickel carbonyl intoxication.

Recently we had the opportunity to record the course of a nickel carbonyl intoxication, and for the sake of interest, and, of course, also because of its

Translated for Information Services Division, EPA, from the Czech by LEO KANNER ASSOCIATES, P. O. Box 5187, Redwood City, California 94063, (415) 365-3046, September 1972.

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relatively rare appearance, we would also like to acquaint interested specialists with it.

In the case mentioned above, we were dealing with a 30 year old chemical engineer, who, as the project leader of the production department of one of the Ostrava chemical plant, was assigned the task of developing tetracarbonylnickel production.

For this purpose, liquid tetracarbonylnickel was first obtained in the first stage through the action of carbon monoxide on metallic nickel at an approximate pressure of 100 atm. The operation was carried out in reactors placed in an open space, and lasted for about 8 hours; of course our patient participated in it directly only for several minutes. The nickel carbonyl which was obtained was conducted through coolers to a glass flask submerged in water, and, after the water has been removed, the engineer mentioned above again filtered, in open space, the nickel carbonyl obtained in the amount of approximately 10 liters, using fritted filters. The filtering itself lasted approximately 5 minutes.

On that day, the engineer finished the work shift without subjective difficulties; however, 6 hours later he had a feeling that there was mucus in his chest. He tried to get rid of it by coughing and inhaling chamomile, but, however, without noticeable effect. At night he slept well. Although the difficulties continued the next day, he did not pay much attention to them and started work as usual. On that day he again made contact with nickel carbonyl. In the laboratory, for a period of about 6 hours, he mixed liquid nickel carbonyl with methyl alcohol in the hood, then added to the mixture acetylene, and then again distilled the nickel carbonyl in the hood. In the process he poured the nickel carbonyl approximately 3 times from a large flask to a smaller flask, which was done outside the hood, and each pouring lasted approximately 2 to 3 minutes.

In the evening after work, the aforementioned difficulties occurred again in intensified form, especially the mucous feeling in the chest, and, in addition to these, an irritation which lead to coughing.

On the third day, in the morning, there was already a pronounced shortness of breath after the slightest effort, and even walking at home in the apartment caused great difficulties. The man stated that he could not inhale as much as he needed, and every attempt to inhale more brought on coughing and pain behind the chest bone. Subsequently the coughing quickly became more and more persistent, and occurred in attacks which lasted approximately 10 minutes. After several hours the temperature was slightly raised. Subsequently the man did not begin work, and was examined at the plant, and, after calcium was administered, he had a feeling of weakness and faintness. He went home and stayed in bed; however, the shortness of breath and the coughing became more intense, and, in the late evening hours, the patient had to brought to the internal disease section in the hospital.

After he was received at the internal disease section, 120/min. tachycardia, slightly raised blood pressure to 150/80, and a pronounced i n s p i r a t o r y d y s p n e a were found with faster respiration. The ER sedimentation was not raised, the blood picture was within the physiological limits, and a urine and sediment analysis also did not show pathological deviations. Oxygen, lobelin, and, later, also glucose were applied.

The skiascopic examination which was made the next morning revealed on the right, near the second rib, a thicker interlobium, and, above it, a shading of bronchiopulmonary origin. The general state of health of the patient gradually improved, and four days later, after the patient was admitted to the hospital, the skiascopic examination no longer revelaed the shading described earlier. From the lung skiagram made at that time, instead of the shading which has been

described, there was only a slightly more pronounced drawing. After 5 hospitalization days, the patient was released and treated at home where he stayed in bed another week.

On the basis of the report about an industrial intoxication, the patient was examined 12 days after release from hospital in our section. Except for pains in the small back, and substernal pains during the objective clinical examination, and respiratory arrhythmia, there were no abnormal findings.

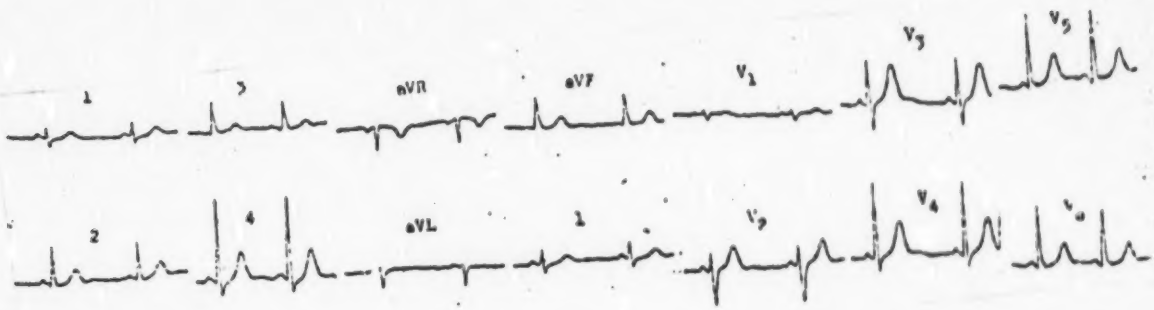
The lung skiagram, except for a very minute calcification in the first right intercostal with a slightly more pronounced bronchiovascular drawing, gave a normal finding. Auxiliary laboratory examinations (blood picture, urine analysis ER sedimentation, and routine tests of the liver) also revealed no pathological changes.

The electrocardiogram examination, which was carried out with standard leads and a series of leads on the extremities and the chest, did not record pathological curves in the recordings. [Graph 1].

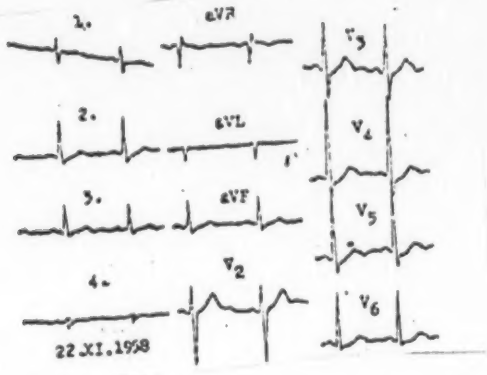
However, we requested the electrocardiogram recordings from the period when the patient was hospitalized. We found, on the electrocardiogram recording which was made on the first hospitalization day [Graph 2], clear signs of ischemic myocardial damage which was marked primarily by a reduced SP sector in the second and third standard leads, and in the third through fifth chest leads, and in the unipolar leads of the extremities (aVK, aVF), while a regular sinus rhythm was recorded with a frequency 100/min, and the intervals were PQ = 0.18, QRS = 0.08.

The electrocardiogram which was made on the fourth hospitalization day [Graph 3] showed still a lower ST_2 and T_3.

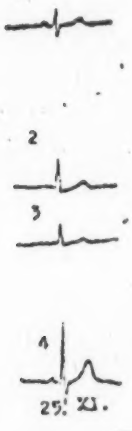
During the control examination which was made in our section four months after the intoxication, the complete finding was normal.



Graph 1



Graph 2



Graph 3

Hence, in the case described, we had an acute tetracabonylnickel intoxication in a laboratory worker after relatively short exposure; the direct contact lasted only several minutes in the two days, if we don't consider the work in the hood on the second day,which lasted approximately 6 hours. Of course, so far, the maximum admissible concentration is unknown; Jacobs [see 9] states it is 0.007 mg/l., and, according to Lazarev [9], intoxications can be caused by concentrations from 7 mg/l.

The first,less serious difficulties occurred in our patient after 6 hours, and appeared as irritations of the upper respiratory tract. After a longer exposition, after 24 hours, the first pronounced picture of heavy i n s p i r a t o r y dyspnea, accompanied by asthmatic cough and substernal pain, appeared. Gradually the clinical picture deteriorated, until, finally, it led to bronchopneumonia and symptoms of temporary ischemic myocardial damage.

The case of the intoxication by carbonylnickel, which we described is very similar to the case described by Moeschlin [10], who also observed, in a laboratory worker, after short exposure, the formation of bronchopneumonia and temporary, but less significant, changes in the electrocardiogram. Also, the subjective difficulties which we found, primarily the inspiratory dyspnea, with retrosternal pains and coughing irritations, agree with the experience of other authors [3,5,7,8,9,10,11]. We did not observe the vertigo [3,5,7,9,10], the headaches [3,5,8,8-10], or the profuse sweating [10] which are often described.

Moreover, bronchopneumonia during nickel carbonylintoxication is described relatively often [5,8,10,14] and often even edema of the lungs is found [4-11]; the finding of myocardial damage during this intoxication is more rare [10].

Neither did we observe the cyanosis which is also often described, the extent of which many authors consider as an indicator of the seriousness of the

intoxication [3,5,7,9-12]. We also did not observe the symptoms of liver damage [9, 10,14] or the central nervous system [5,7,9,10] which are sometimes quoted.

The mechanism of the toxic nickel carbonyl effect seems so far not completely known [7,8,9], however, it is considered to be certain that the formation of methemoglobin does not play an important role [6]. Most authors [7,9,10,11] are of the opinion that nickel carbonyl acts in the organism as a whole molecule with its powerful reduction effect as a catalytic poison, which affects the system, especially the nerves and the entire metabolism. Amor [see 11] explains the effect of nickel carbonyl by the dissociation effect of nickel and liberated CO; acccrding to Vahlen [see 4], nickel carbonyl dissolves in the lungs into carbon monoxide and other gas nickel compounds which are more toxic than CO. Similar mechanisms could, perhaps, possibly explain Moeschlin's and our finding of the simultaneous damage to the respiratory tracts and the temporary myocardial changes. Barnes [1] presupposes the action of highly active nickel on the enzymes in the endothelium of the capillaries, and proves experimentally that nickel has an affinity to the sulfhydryl cysteine group which reduces its toxicity. It is precisely the effect of metals on the biological system which is attributed to the formation of mercaptides with sulfhydryl groups of protein compounds in the cellular system. In this respect, perhaps a somewhat different opinion about the effect of the BAL antidote may be of interest: while Sunderman et al. [13] obtained positive results using BAL, Barnes [1] proves, through the results of his experimental tests, the low BAL effectiveness. However, in their reports, the authors did not come to terms with the complex formation index of this substance on nickel. Brandes [see 11] compares the effect of nickel carbonyl with the effect of phosgene.

From the numerous data from the literature about the pathological-anatomical changes caused by nickel carbonyl intoxication, it is evident that it has an effect on the damage to the epithelium of the respiratory tract which is similar to the changes caused by the influenza virus [7,10], oversaturation and bleeding in the lung parenchyma and, finally, the destruction of the lung parenchyma and epithelium [11], and tiny hemorrhages in the white brain matter [4,9]. Sometimes, also, inflammation changes on the mucous membranes of the stomach and intestine are reported [7,9].

Summary

The authors describe an acute tetracarbonylnickel intoxication by inhalation which developed in a laboratory worker after brief exposure.

After an initial irritation of the upper respiratory ducts, signs of respiratory dyspnea and a suffocating cough developed, which were accompanied by substernal pain leading to bronchopneumonia and temporary ischemic myocardial damage.

The authors compare the intoxication symptoms which were detected with data about the clinical picture and the pathogenesis of nickel carbonyl intoxications described in the literature.

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Submitted for publication on March 24, 1959.